This presentation describes current knowledge about the treatment of patients with the dual diagnosis of post-traumatic stress disorder and substance misuse, a population that is typically considered ''difficult to treat''. We will cover background on PTSD and substance misuse (including rates, the typical client, models and stages of treatment, and clinical dilemmas). Clinical interventions will also be addressed, with a focus on "Seeking Safety", an evidence-based model for PTSD and substance misuse.

Symposium: Recent advances in the understanding of adolescent conduct disorder

S59.01

Emotional processing in conduct disorder: Data from psychophysiology and neuroimaging

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Per definition adults with antisocial personality disorder have met the diagnostic criteria of conduct disorder since adolescence at least. Those who have started to show antisocial behavior before the age of ten tend to exhibit even more severe aggressive behavior throughout early adulthood. However, conduct disorder is not a homogenous category that is characterized by a steady development towards a specific antisocial disorder in adulthood. Within the category of conduct disorder there are particularly high differences in emotional processing and an individual's capability to regulate emotions. Regarding the neurobiological correlates of emotional processing in subjects with conduct disorder, psychophysiological data suggest high stability over time with the main finding of hypoarousal in resting states and autonomous hyporeagibility to emotional stimuli. Findings from functional neuroimaging are more heterogeneous probably reflecting subtypes of antisocial disorders which are subsumed under the category of conduct disorder. Developmental research in psychiatry needs a multidimensional diagnostics including a precise psychopathological characterization and subdifferentiation. Although genetic dispositions are stable, they interact with changing psychosocial factors which take influence on the developing brain in more or less vulnerable stages.

Symposium: Vulnerability factors in depression across the life span

S04.01

Genetic determinants of neurobiological vulnerability markers in depression

M. Ising [1], S. Adena [1], E. Binder [1], A. Pfennig [1], M. Schalling [2], B. Mueller-Myhsok [1], S. Modell [1], F. Holsboer [1]. [1] *Max-Planck-Institute of Psychiatry, Munich, Germany* [2] *Department of Molecular Medicine, Karolinska Institute, Stockholm, Sweden*

Background and Aims: Neuroendocrine changes of the stress hormone system and REM sleep abnormalities are potential vulnerability markers for depression. Investigating underlying genetics provides new insights into the molecular pathways of these endophenotypes and into the pathophysiology of depression vulnerability. We selected a high-risk linkage and endophenotype approach, i.e., we investigated REM sleep abnormalities (REM density) and altered stress hormone regulation (dex/CRH test) in families with a high prevalence of major depression.

Methods: Eleven families were so far included, comprising 82 high risk family members. 32 of them were unaffected, 33 remitted, and 17 suffered from an affective disorder at the time of the investigation. Illumina Infinium Whole Genome genotyping with 100k bead chips was performed. Variance component (VC), and a quantitative trait linkage analysis (QTL) on polysomnographic (REM density) and neuroendocrine vulnerability markers (dex/CRH test) were applied.

Results: Linkage analysis (VC, QTL) revealed suggestive linkage (LOD score > 2) for altered REM density at loci of the chromosomes 2, 4, 8 and X. The linkage results on chromosomes 2 and X correspond to previous findings. No results were obtained with a classical diagnosis based linkage approach.

Conclusions: The use of quantitative vulnerability markers in a high risk family study and a SNP based whole genome approach revealed a number of loci with suggestive linkage, that were not detectable with the classical linkage approach. Our findings suggest the suitability of investigating vulnerability marker in combination with a SNP based whole genome approach in complex disorders like depression.

S04.02

Psychological vulnerability factors and neuroendocrine and sleep regulation in healthy children and adolescents

S. Brand [1], J. Beck [1], F. Muheim [1], M. Hatzinger [2], E. Holsboer-Trachsler [1]. [1] *Psychiatric University Clinics, Depression Research Unit, Basel, Switzerland* [2] *Psychiatric Out-Patient Department, University Hospital Basel, Basel, Switzerland*

Background and Aims: The presentation aims at summarizing current knowledge about sleep in children and adolescents and at describing possible factors influencing their sleep.

For preschoolers, there is evidence that objectively assessed (sleep-EEG, actigraphy) poor sleep is associated with increased endocrine activity; this is to say, with increased morning cortisol secretion, an associative pattern observed so far only in adults. Furthermore, poor sleep and increased cortisol secretion are associated with emotional and behavioral difficulties.

During life span, notable changes occur with respect to sleep quantity and quality. Compared to childhood, in adolescence, three prominent changes occur: First, sleep quantity declines from about 10 hours at 10 years of age to between 6.5 and 8.5 hours in older adolescents. Second, a marked shift towards a longer sleep duration and later bed time from school nights to weekend nights is observable. Third, daytime sleepiness (20%) and insomnia symptoms (25%) are common among adolescents.

Among a variety of factors affecting adolescents' sleep, we could show that negative parenting styles unfavorably influenced adolescents' sleep quality, suggesting that even 18 years old adolescents may be far away from been emotionally independent from their parents. Furthermore, the so-called weekend-shift was correlated with increased sleepiness during the week, suggesting that irregular sleep schedules may negatively influence sleep quality and daytime functioning.

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Last, if compared to healthy controls, children and adolescents after cleft lip and palate (CLP) repair were not at risk reporting sleep difficulties; rather, irrespective of the presence of CLP, sleep was affected by psychological strain.

S04.03

Sleep regulation and cognitive performance in elderly subjects with dementia and depression

U. Hemmeter [1], R. Rocamora [3], A. Thum [1], A. Haag [1,2], M. Hatzinger [5], W. Cassel [4], C.J. Krieg [1], E. Holsboer-Trachsler [5]. [1] *Clinic of Psychiatry and Psychotherapy, University of Marburg, Marburg, Germany* [2] *Clinic of Neurology, University of Marburg, Marburg, Germany* [3] *Hospital Psiquiatric Universitari Institut Pere Mata, Reus, Spain* [4] *Clinic of Internal Medicine, Sleep Laboratory, University of Marburg, Marburg, Germany* [5] *Depression Unit, Psychiatric University Clinics (UPK) Basel, Basel, Switzerland*

In elderly patients who suffer from depressive symptoms and cognitive impairment the clinical decision between the diagnoses of depression and dementia may be difficult. In addition, patients with dementia and depressed patients frequently show a disturbance of sleep. Sleep EEG registration in depression revealed a characteristic sleep EEG profile concerning distinct alterations of sleep architecture and REM-sleep (reduction of SWS, increase and advance of REM-sleep). In dementia polysomnographic assessment has been done less intensively, mainly in patients with dementia of Alzheimer type (DAT). The most significant polysomnographic finding in DAT is a reduction of REM-sleep, which may reflect impaired cholinergic neurotransmission. Therefore, predominantly REM-sleep variables clearly differ between depressed patients and patients with DAT.

In this presentation polysomnographic data and data of cognitive performance in dementia and depression will be reviewed. In addition, own long term studies in patients with different types of dementia and in depressed patients will be presented. The polysomnographic findings of these studies will be discussed with respect to differential diagnosis, prediction of treatment response and the long term course of both diseases. In addition, the results will be related to the current knowledge of the neurochemical and neuroendocrine regulation of sleep.

S04.04

Neuroendocrine and sleep regulation as predictors of illness course and therapy in depression

M. Hatzinger [1], S. Brand [2], U. Hemmeter [3], M. Ising [4], E. Holsboer-Trachsler [2]. [1] *Psychiatric Out-Patient Department, University Hospital Basel, Basel, Switzerland* [2] *Depression Research Unit, Psychiatric University Clinics Basel, Basel, Switzerland* [3] *Psychiatric Department, University of Marburg, Marburg, Germany* [4] *Max Planck Institute of Psychiatry, Munich, Germany*

Background and Aims: In depression, changes in EEG sleep measures are well documented findings. However, the predictive value of these alterations for treatment and long-term course of depression still warrants clarification. Therefore, we examined whether the previous course of depression, treatment response during antidepressant therapy, and the long-term outcome in follow-up are associated with sleep regulation. Since the hypothalamic-pituitary-adrenocortical (HPA) system may play a crucial role in depression's neurobiology, we evaluated HPA system function as well.

Methods: 15 patients (4 men, 11 women; age 43–59) with depression were enrolled in the study. HPA system assessment using the combined DEX/CRH test and sleep EEG studies were conducted at baseline, after a 6 week antidepressant treatment period (trimipramine), and at follow-up, i.e., after 2-10 years.

Results: The previous clinical course, i.e., the number of episodes until baseline, correlated significantly with EEG sleep measures i.e. sleep continuity values, slow wave sleep (SWS) and REM latency.

During treatment sleep continuity values improved and the correlation with the previous long-term course disappeared. The correlation with SWS persisted. The only sleep EEG marker at baseline predictive for treatment response was REM latency.

In the prospective long-term outcome SWS and REM density variables were related to the occurrence of recurrences. These sleep EEG markers correlated closely with HPA system regulation.

Conclusions: The long-term outcome of depression is related to the sleep EEG pattern: SWS and REM density measures may reflect predictive markers for the long-term course. These markers are associated with HPA system regulation.

Plenary Lecture: Pathways to integrative care in childhood

PL01.01

Pathways to integrative care in childhood

S. Kutcher [1,2]. [1] *Sun Life Financial Chair in Adolescent Mental Health, Dalhousie University, Halifax, NS, Canada* [2] *WHO Collaborating Center in Mental Health and Training and Policy Development, Dalhousie University, Halifax, NS, Canada*

Mental health care has traditionally been provided within the framework of a verticle mental health services system. Additionally, mental health care is often provided through a variety of agencies or organizations that have a number of different mandates, responsibilties, authorities and accountabilities. These are not well linked with or to each other.

Furthermore, mental health services for children and youth are often not well integrated into adult mental health services. This profusion of confusion regarding mental health care for young people can be much better defined and operationally developed if a model of care based on population mental health care needs and provider mental health care competencies can be applied. This presentation will present the conceptual framework for such a model that allows for mental health care to be intergrated into all levels of health and other systems that provide interventions for young people suffering from a variety of mental health problems.

State-of-the-art Lecture: Physical illness in persons with severe mental disorders

SOA01.01

Physical illness and access to medical services in people with schizophrenia

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